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     (FILE 'HOME' ENTERED AT 09:43:04 ON 16 FEB 2004)

     INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI,
     BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA,
     CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DISSABS,
     DDFB, DDFU, DGENE, DRUGB, DRUGMONOG2, ...' ENTERED AT 09:43:33 ON 16 FEB
     2004
                SEA HUMAN KINASE
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                 1   FILE BIOCOMMERCE
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               104   FILE BIOTECHABS
               104   FILE BIOTECHDS
                30   FILE BIOTECHNO
                 1   FILE CABA
                15   FILE CANCERLIT
               155   FILE CAPLUS
                 2   FILE CEABA-VTB
                 4   FILE CIN
                 7   FILE DISSABS
                 1   FILE DDFU
              2145   FILE DGENE
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                25   FILE TOXCENTER
               261   FILE USPATFULL
                49   FILE USPAT2
               149   FILE WPIDS
               149   FILE WPINDEX
L1               QUE HUMAN KINASE
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                 SEA F5-F20
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                0*   FILE DGENE

     FILE 'CAPLUS, WPIDS, BIOSIS, BIOTECHDS, USPAT2, MEDLINE, SCISEARCH,
     EMBASE, BIOTECHNO, ESBIOBASE, LIFESCI, TOXCENTER, CANCERLIT, PROMT'
     ENTERED AT 09:46:28 ON 16 FEB 2004
L2           521 S L1 AND (INHIBIT? OR MODULAT? OR AGONIST OR ANTAGONIST)
L3           152 S L2 AND AGONIST
L4            98 DUP REM L3 (54 DUPLICATES REMOVED)
L5           175 S L2 AND ANTAGONIST
L6           112 DUP REM L5 (63 DUPLICATES REMOVED)

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L4 ANSWER 96 OF 98 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:640772 CAPLUS
DOCUMENT NUMBER: 127:304772
TITLE: A kinase capable of site-specific phosphorylation of IκB-α and its regulation and use in the diagnosis or treatment of NF-κB-related diseases
INVENTOR(S): Chen, Zhijian J.
PATENT ASSIGNEE(S): Proscript, Inc., USA
SOURCE: PCT Int. Appl., 141 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9735014	A1	19970925	WO 1997-US4195	19970319
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2249551	AA	19970925	CA 1997-2249551	19970319
AU 9725317	A1	19971010	AU 1997-25317	19970319
AU 728679	B2	20010118		
EP 910645	A1	19990428	EP 1997-916786	19970319
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
BR 9710408	A	19990817	BR 1997-10408	19970319
JP 2000510328	T2	20000815	JP 1997-533584	19970319

PRIORITY APPLN. INFO.: US 1996-616499 A 19960319
WO 1997-US4195 W 19970319

AB A large multi-subunit protein kinase that catalyzes the site-specific phosphorylation of the NF-κB **inhibitor** IκBα after activation by ubiquitinylation by E1 and E2 enzymes is described. The enzyme has 10 subunits of 31, 33, 36, 38, 40, 43, 50, 55, 62, 70, and 85 kilodaltons and phosphorylates IκBα at residues 32 and 36. CDNAs encoding the subunits of the enzyme may be cloned and characterized for use in the manufacture of the enzyme, e.g. for preparation of the enzyme for assays or preparation of antibodies for diagnostic or therapeutic use. The cDNAs may be used to develop probes to detect and quantify gene expression or in gene therapy. **Agonist** or **antagonist** ligands of the kinase may be of diagnostic or therapeutic use. More specifically, this invention relates to selective **inhibitors** of the kinase and to structure-based design of ligands, **agonists**, and **antagonists** of the kinase.

L4 ANSWER 97 OF 98 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:253031 CAPLUS
DOCUMENT NUMBER: 118:253031
TITLE: Stimulation of MHC class I transcription by interferon-γ involves a non-A, non-C kinase in addition to protein kinase C
AUTHOR(S): Radford, James E., Jr.; Waring, Jeffrey F.; Pohlman, Joyce K.; Ginder, Gordon D.
CORPORATE SOURCE: Inst. Hum. Genet., Univ. Minnesota, Minneapolis, MN, 55455, USA
SOURCE: Journal of Interferon Research (1993), 13(2), 133-41
CODEN: JIREDJ; ISSN: 0197-8357
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The signal pathways by which interferon-γ (IFN-γ) is able to up-regulate major histocompatibility complex (MHC) class I transcription were studied in two human hematopoietic tumor cell lines, K562 and Ramos. These studies suggest that the IFN-γ signal is transduced via an H 7- and staurosporine-sensitive kinase that is distinct from protein kinase C (PKC) and protein kinase A (PKA) in both cell types. Ramos cells appear to utilize an addnl. pathway involving double-stranded RNA-dependent protein kinase. PKC and possibly PKA appear to be involved in one or more intersecting pathways by which **agonists** of these kinases are able to act synergistically with IFN-γ, but activation of these latter pathways is neither necessary nor sufficient for induction of MHC class I transcription. **Modulation** of G-protein- and Ca2+-calmodulin-associated pathways and arachidonic acid metabolism had no effect on constitutive or IFN-γ-stimulated class I transcription. The class I stimulatory factor produced in response to IFN-γ treatment appears to have a short t1/2. The identity of this factor is unknown, but is likely to be distinct from known mediators of IFN-stimulated transcription. Gene and cell-type specificity in the signal transduction pathways utilized by IFN-γ implies that such pathways may be useful targets for exptl. and therapeutic manipulation.